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A TENTATIVE ESTIMATION OF MAN'S TOLERANCE TO OVERPRESSURES FROM AIR BLAST

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and

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ABSTRACT

Tentative estimates of the "sharp"-rising overpressures as a function of duration which represent a lethal hazard to the 70-kg animal 1, 50 and 99 per cent of the time were presented. The predictions were based on interspecies correlations and extrapolations encompassing blast-tolerance data for six mammalian species. The tentative application of the data to indicate human blast tolerance was discussed and relevant uncertainties in the estimates were emphasized. It was also pointed out that biologic tolerance would be different for air-blast pulses having non-ideal wave forms frequently associated with various geometries of exposure.

Selected pathophysiological information pertinent to the biological response following blast exposure was given; namely, survival time and selected postshot observations of dogs and goats.

Experiments reported herein were conducted according to the "Rules Regarding Animal Care" established by the American Medical Association.

INTRODUCTION

Biological effects of air blast are commonly segregated arbitrarily into three categories: (1) primary effects—those caused by the overpressure itself; (2) secondary effects—those due to the impact of objects, including fragments of the explosive container if any, set in motion by the blast wave; and (3) tertiary effects—those associated with displacement of a biological target, be they accelerative or decelerative in character. This presentation deals only with the primary effects of blast.

The purpose of this report is fivefold: first, to present selected experimental data useful in relatively assessing blast tolerance among six mammalian species of animals exposed to "sharp"-rising overpressures of various durations; second, to set forth one analytical procedure for extrapolating the data to predict tolerance for a 70-kg mammal; third, to formulate tentative estimates for human tolerance to ideal or near-ideal wave forms; fourth, to describe experiments showing that variations of the geometry in which exposure to air blast occurs can alter the effect quantitatively from that expected in "free-field" situations; and finally, to present lethality-time data and selected postshot observations of animals along with the lesions believed responsible for the observed effects.

LETHALITY DATA FOR EXPERIMENTAL ANIMALS

In previous reports,[1-4] experiments were described wherein experimental animals were exposed to "sharp"-rising overpressures produced by a variety of shock tubes (1685 animals) and by high-explosive charges of different magnitudes (993 animals). Subsequently, additional dog and goat studies, as yet unpublished, were carried out with high explosives and shock tubes (296 animals to date). In all instances the tolerance of the animal was assessed using lethality as an end point; also, the overpressures associated with 50 per cent lethality (LD_{50}) were computed from probit curves relating per cent mortality to the magnitude of the overpressures for pressure pulses of several durations.

The data are assembled in Figure 1 which shows the overpressures producing lethality in 24 hours for six different species of animals as a function of pulse duration ranging from about 0.4 to 7000 msec. Attention is directed to the fact that, though the curves for each animal species are flat for the longer-duration overpressures, they each rise progressively for the shorter-duration pulses; i.e., there is for each species a critical duration longer than which the lethal overpressure remains fairly constant and, shorter than which, it rises significantly. Also, it is clear that while the data generally show the larger

OVERPRESSURE FOR 50 PER CENT LETHALITY
AS A FUNCTION OF DURATION

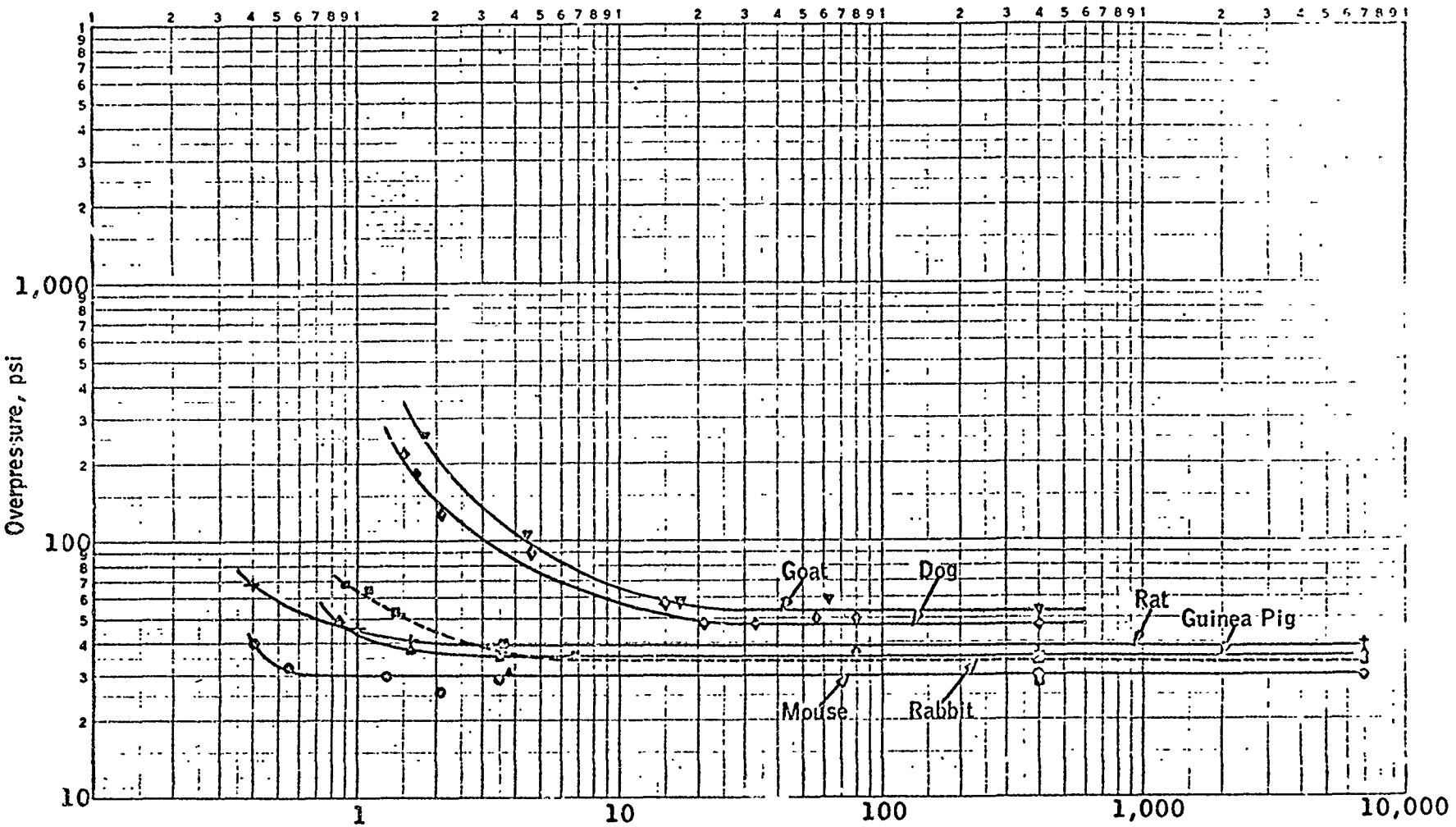

Figure 1

animals are more tolerant to blast overpressures — especially for the shorter-duration pulses, there specifically exists no completely uniform relationship with body size except possibly at the extreme left side of Figure 1; viz., for very short-duration blast waves.

EXTRAPOLATION OF DATA

The LD_{50} Values

Using the experimental points in Figure 1 for the pulses of 400 msec duration, the LD_{50} overpressures for each species of animal were, in a previous study,[3] plotted against the average body weight, as shown in Figure 2. A regression curve, fitted by the least squares method and showing a standard error of the estimate of 13.9 per cent, was solved to obtain an extrapolated LD_{50} figure of 50.5 psi for a mammal weighing 70 kg.

A similar exercise to obtain LD_{50} values for the 70-kg mammal applicable to pulse durations of 3, 5, 10, 30, 60 and 400 msec was carried out in this study, except that the LD_{50} numbers for each species at the pulse durations of interest were read from the smoothed curves shown in Figure 1. The results are given in Table 1 and shown graphically by the middle curve labeled LD_{50} in Figure 3.

The LD_1 and LD_{99} Values

To obtain an estimate of the LD dose for 1 and 99 per cent lethality applicable to the 70-kg animal, a probit regression equation was used having the following form:

$$y = a + b \log x$$

y = per cent mortality in probit units

x = pressure dose, psi

a = intercept constant

b = slope constant

Given the LD_{50} values for the 70-kg mammal shown in Table 1, one has y = 5 (50 per cent mortality) and x = the LD_{50} pressure. It is also necessary to have a slope constant, b, for the equation to allow solving for a, the intercept value. After this, one may solve the probit equation obtained by substituting desired values for y and

RELATION BETWEEN BODY WEIGHT AND FAST-RISING OVERPRESSURES OF 400 MILLISECONDS DURATION NEEDED TO PRODUCE 50 PERCENT MORTALITY

Animals exposed side-on against the plate closing the end of a shock tube

REGRESSION EQUATION

$\text{Log } (LD_{50}) = 1.3673 + 0.06939 \log (BW)$

Where LD_{50} = Pressure required for 50% mortality, psi

BW = Average body weight of the group, grams

Standard Error of Estimate: 0.0602 log units (13.9 %)

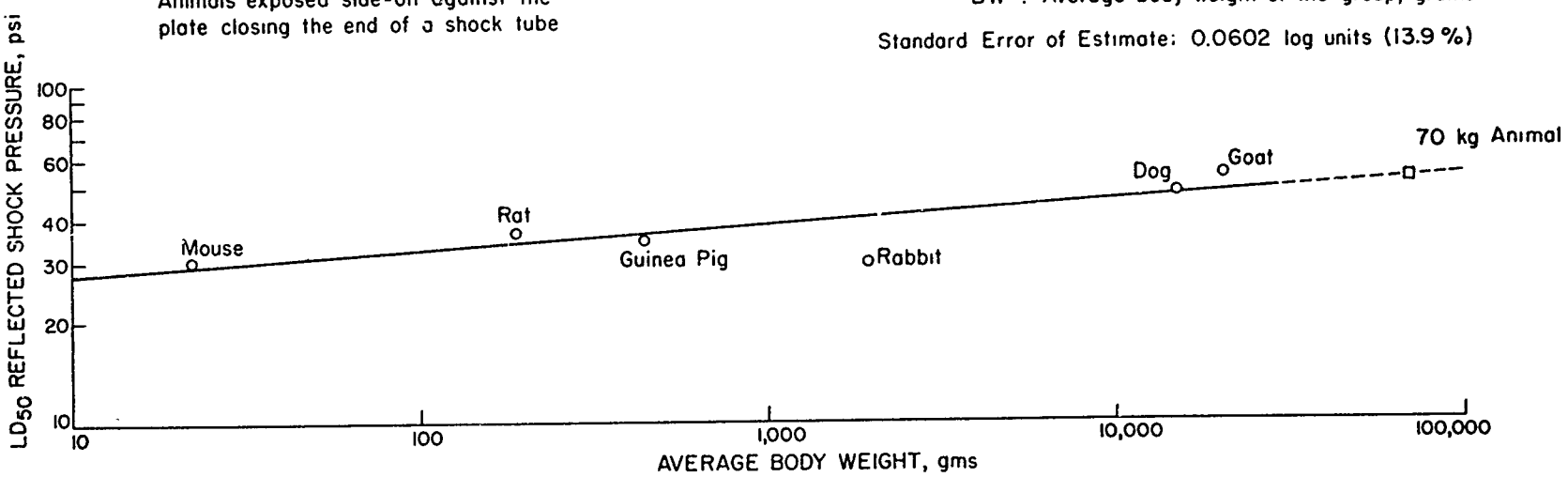

Figure 2

Table 1

RELATION BETWEEN "SHARP"-RISING OVERPRESSURE AND PULSE DURATION REQUIRED FOR 50-PER CENT LETHALITY

Species	Mean Body Weight	LD_{50} Pressures in psi*					
		Duration, msec 400	60	30	10	5	3
Mouse	22.00 g	29	29	29	29	29	29
Rat	192.00 g	36	36	36	36	36	36
Guinea Pig	445.00 g	34	34	34	34	34	34
Rabbit	1.97 kg	33	33	33	33	33	38
Dog	16.50 kg	49	49	49	60	80	106
Goat	22.20 kg	53	53	53	68	96	138
Mammal	70.00 kg	52	58	64	98	185	431

*All the LD_{50} values were picked from the curves in Figure 1 except those for the 70-kg animal which were calculated.

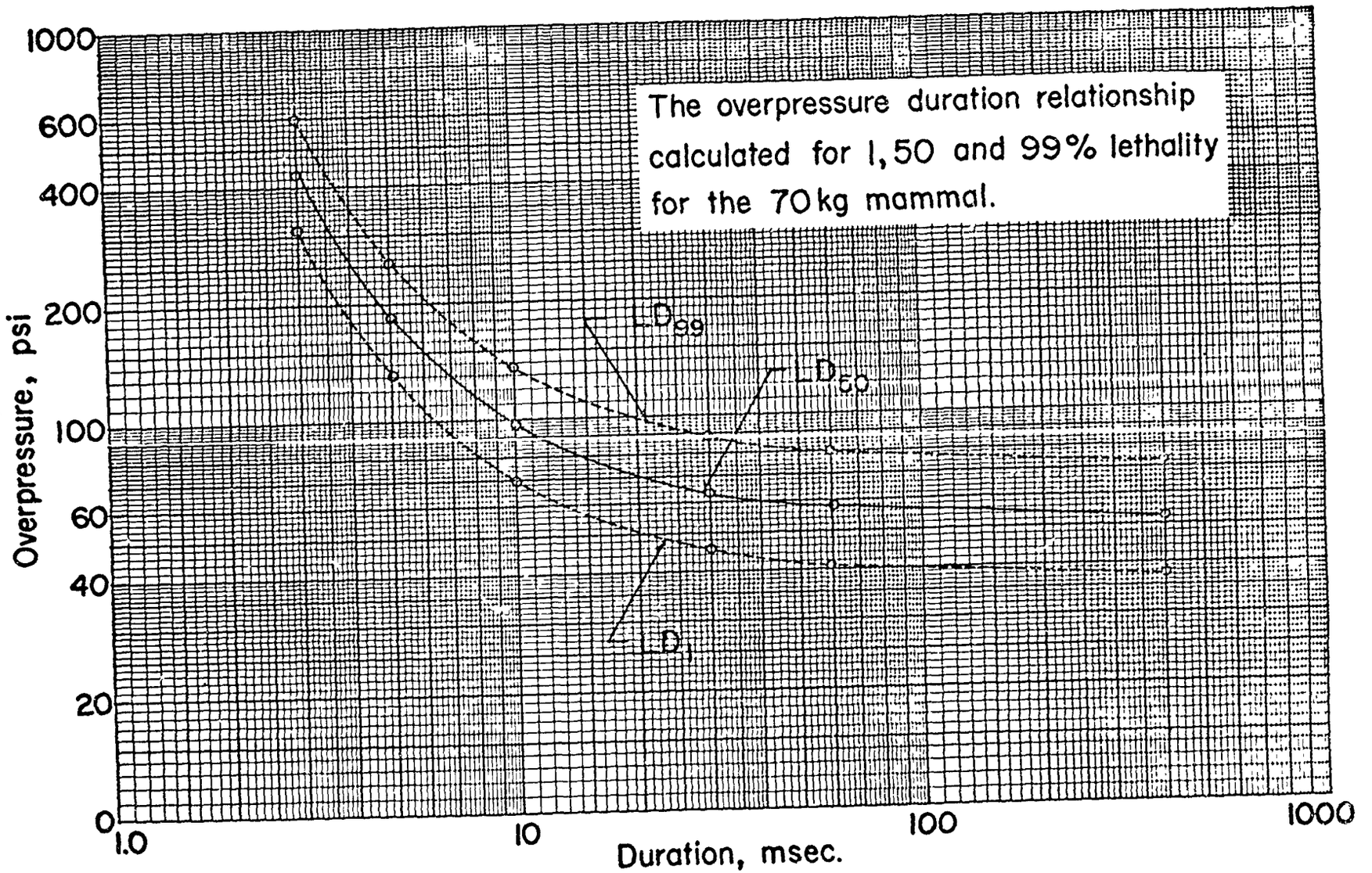
The overpressure duration relationship calculated for 1, 50 and 99% lethality for the 70 kg mammal.
LD99
LD50
LD1
Overpressure, psi
1000
600
400
200
100
60
40
20
0
1.0
10
100
1000
Duration, msec.

Figure 3

computing the x or overpressure figures.

Though there is no proven justification for believing that the slope constant for the 70-kg animal is the same for all durations of overpressure nor for being absolutely sure that there is one value for the slope constant for all the animals studied, the assumption that these contingencies were so was made here and an average value of b = 16.151* was used to solve probit regression curves for the extrapolated results which allowed calculation of the LD_1 and LD_{99} overpressures. The use of the procedure yielded the results given by the lower and upper curves of Figure 3, respectively.

There is specifically one study[4] involving the four smaller species of animals in which the slopes of the probit regression equations applicable to each type of experimental animal were tested for parallelism. Their slopes were not significantly different from one another. If one averages the adjusted slopes for each species, one obtains b = 15.371, a value probably not at variance with the figure b = 16.151 mentioned above. Another study[3] also reported that probit regression curves for six species of animals were essentially parallel. The adjusted slope for the entire series (569 animals) was 17.159, also a figure for b very close to that employed here.

ESTIMATION OF HUMAN BLAST TOLERANCE

One must approach the use of the extrapolated animal data to predict human tolerance to blast overpressures with considerable caution for many reasons. Several will be mentioned here. First, the animal data presented above apply only to "fast"-rising overpressures involving ideal or near-ideal wave forms. Therefore, any application of the figures for the 70-kg mammal to man needs be strictly limited to classical or near-classical wave forms, a fact that will be emphasized later.

Second, a glance at Figure 2 giving the 400-msec data shows that some animals are above and others below the regression curve. One wishes to know whether human tolerance in truth is above or below the "average" animal data and what are the quantitatively applicable figures. Unfortunately, such numbers are currently unavailable.

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*Obtained by adjusting all the dog and goat probit mortality curves parallel.

Third, for the shorter-duration overpressures, the lethality curves progressively rise and any estimates of tolerance would appear to be more uncertain than is the case for the longer-pulse durations for which the lethality-duration curves are flat.

Fourth, the animal data apply to lethality assessed over a 24-hour period. Information applicable to the guinea pig is at hand[5] which shows that death from blast exposure may occur as late as the 17th post-exposure day. Too, it is known that death following exposure to blast can be delayed in man.[6]

On the more positive side, three eventualities seem clear, however; namely, (1) that the tolerance of the adult human is not likely to be too far from the results obtained with six different mammalian species; (2) that the pressure-duration relationship demonstrated in animals also holds for the human case; i.e., quite high overpressures are required for lethality if the pulse duration is "short" and minimal overpressure will kill if the overpressure durations are "long;" and (3) that tentative estimates of human tolerance are justified on the basis of the considerable data at hand provided allowances are made for the many uncertainties that obviously exist.

Accordingly, it is suggested that Figure 3 be taken as a guide and the data adjusted to "fix" a range of figures likely to bracket acute tolerance of adult man to air blast. The suggested adjustment is 10 per cent above and 20 per cent below the mean animal extrapolation presented above. Thus, Table 2 summarizes the arbitrary and tentative LD_{50} figures estimated for "sharp"-rising overpressures of six durations ranging from 3 to 400 msec.

It it well to emphasize here that data are available[7-10] suggesting that the overpressure values shown in Figures 1, 2 and 3 as well as those in Tables 1 and 2 may be incident or reflected shock pressures as long as they are "sharp" rising; i.e., the application of the incident plus the reflected pressure occurs almost instantaneously. For example, a biological target located against a reflecting surface would receive a "sharp"-rising reflected overpressure of 60 psi at the range where an incident shock of 20 psi would occur.

Also to be emphasized is the statement that man's tolerance to air-blast overpressures having other than classical wave forms can be expected to be quite different; i.e., an animal's tolerance to smooth-rising overpressures[10] or those rising in steps sufficiently separated in time is higher than is the case for ideal or near-ideal pressure pulses.[5,6]

Table 2

TENTATIVE ESTIMATE OF PRESSURE-DURATION RELATIONSHIP FOR 50-PER CENT LETHALITY IN ADULT HUMANS (70 kg)*

LD_{50} Pressure psi	Pulse Duration msec	LD_{50} Pressure psi	Pulse Duration msec
42-57	400	78-108	10
46-64	60	148-204	5
51-70	30	345-474	3

*Applies to "sharp"-rising overpressures of ideal or near-ideal wave forms.

"FREE-FIELD" VS. "GEOMETRIC" SCALING

"Sharp"-rising blast waves occurring "free-field" are usually distorted upon passing into structures. The primary blast hazard therefore depends upon the wave form encountered in a given geometry. For instance, dogs tolerated long-duration overpressures of over 100 psi when the pressure rose in a smooth manner and peaked in 30, 60, 90, and 155 msec.[10] Also the resistance of animals to overpressure increased when the latter was applied in two steps.[5,6]

Different species have been exposed to air-blast waves of long duration applied in two steps by mounting them at various distances upstream of a reflecting surface.[1,5-8] The time interval between steps (the time between the passing of the incident shock and the return of the reflected shock) was a function of the animal's distance from the reflecting plate. According to Figure 4, the tolerance of guinea pigs and dogs to overpressure rose when the time between shocks was increased beyond 0.20 msec and 0.40 msec, respectively. There appeared to be a relation between species size and the length of time between shocks associated with increased tolerance.

That the geometry of exposure can have a marked effect on biological response expected in terms of "free-field" pressures can be illustrated in the following experiments. Guinea pigs were exposed to air-blast pulses of long duration while located in shallow, deep, and deep-with-offset chambers mounted on a shock tube.[11] The results given in Table 3 show that the incident shock pressures required to kill 50 per cent of the animals in the deep, deep-with-offset, and shallow chambers were 19.5, 26.8, and 34.9 psi, respectively. Thus, the amount of protection against the blast wave afforded by the chambers may be taken in that order.

Obviously, the shock wave entering the deep and deep-with-offset chambers reflected from the bottom and downstream walls. In so doing, the reflected pressure within these chambers was higher than that in the incident shock (Figures 5 - 7). Since the guinea pigs all but filled the volume of the shallow chambers, significant reflections did not occur and the incident shock pressures were considered to be the "dose" at the animal's location. The pressure "dose" for animals in the deep chamber was taken from gauge "c" in the lateral wall of the chamber (Figure 6); and for animals in the deep-with-offset chambers, the "dose" was taken from gauge "c" in the bottom of the chamber (Figure 7).

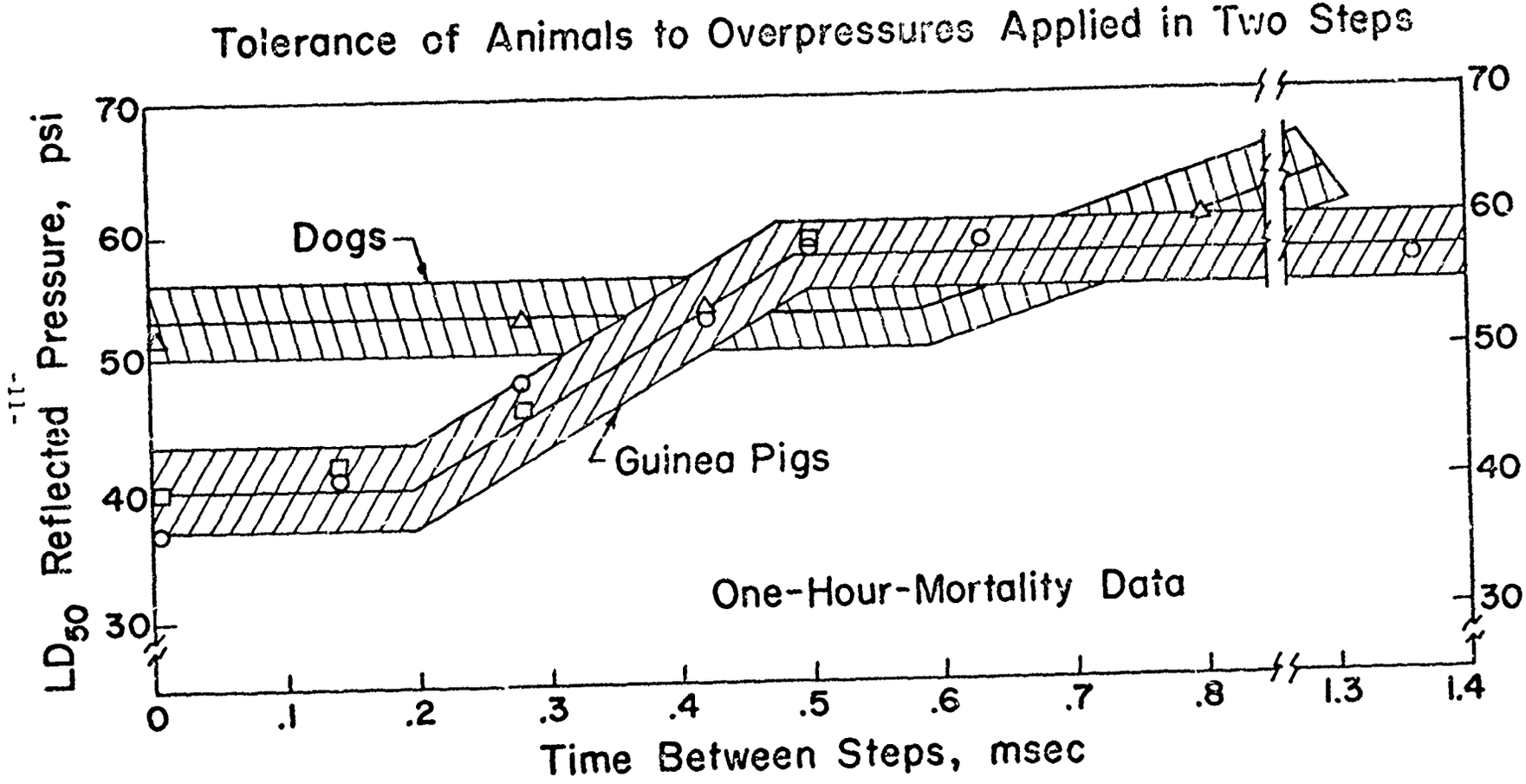

Tolerance of Animals to Overpressures Applied in Two Steps
LD50 Reflected Pressure, psi
70
60
50
40
30
Dogs
Guinea Pigs
One-Hour-Mortality Data
0
.1
.2
.3
.4
.5
.6
.7
.8
1.3
1.4
Time Between Steps, msec

Figure 4

Table 3

RESULTS OF THE PROBIT ANALYSIS RELATING LETHALITY TO OVERPRESSURE*

Chamber Geometry	Number of Guinea Pigs	LD_{50}, psi
Incident Shock		
Shallow	40	34.9 (33.4-47.8)**
Deep	38	19.5 (17.4-21.0)
Deep-with-Offset	40	26.8 (24.2-29.6)
Reflected Pressure		
Deep	38	34.6 (31.5-37.2)
Deep-with-Offset	40	35.9 (33.0-38.8)

*See text for wave forms and durations of the pressure pulse.

**95-per cent confidence limits.

PRESSURE-TIME RECORDS ASSOCIATED
WITH THE SHALLOW CHAMBER

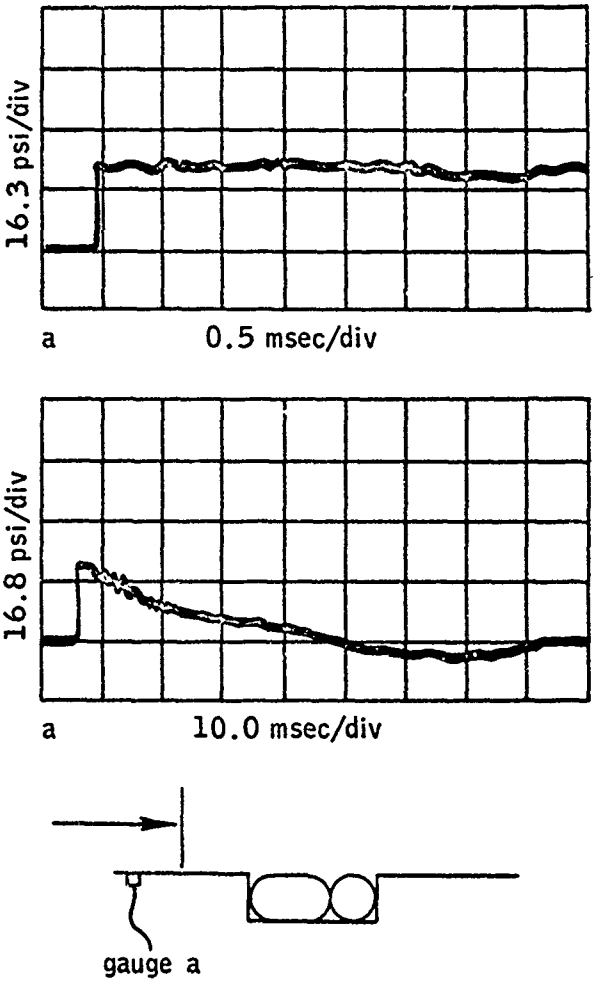


Figure 5

PRESSURE-TIME RECORDS ASSOCIATED
WITH THE DEEP CHAMBER

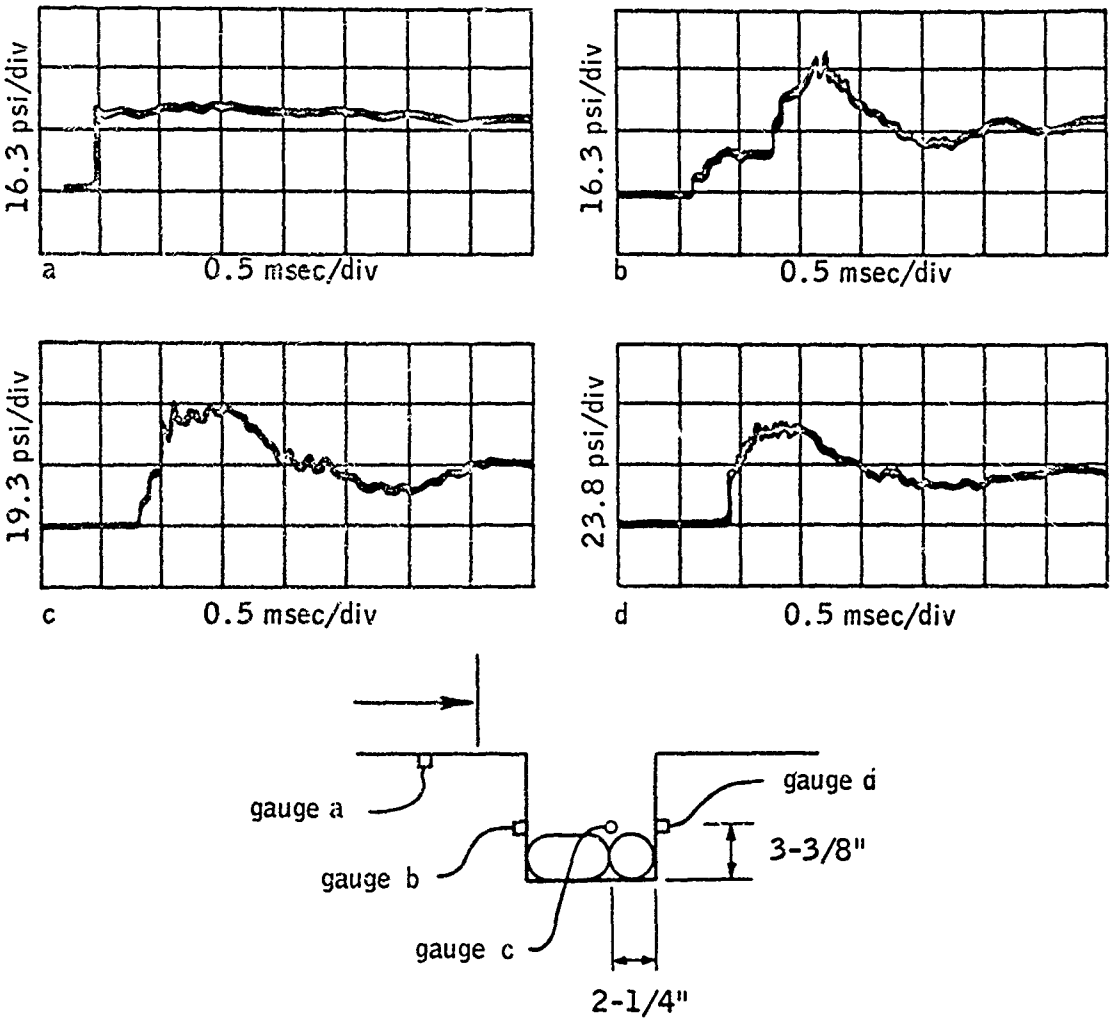
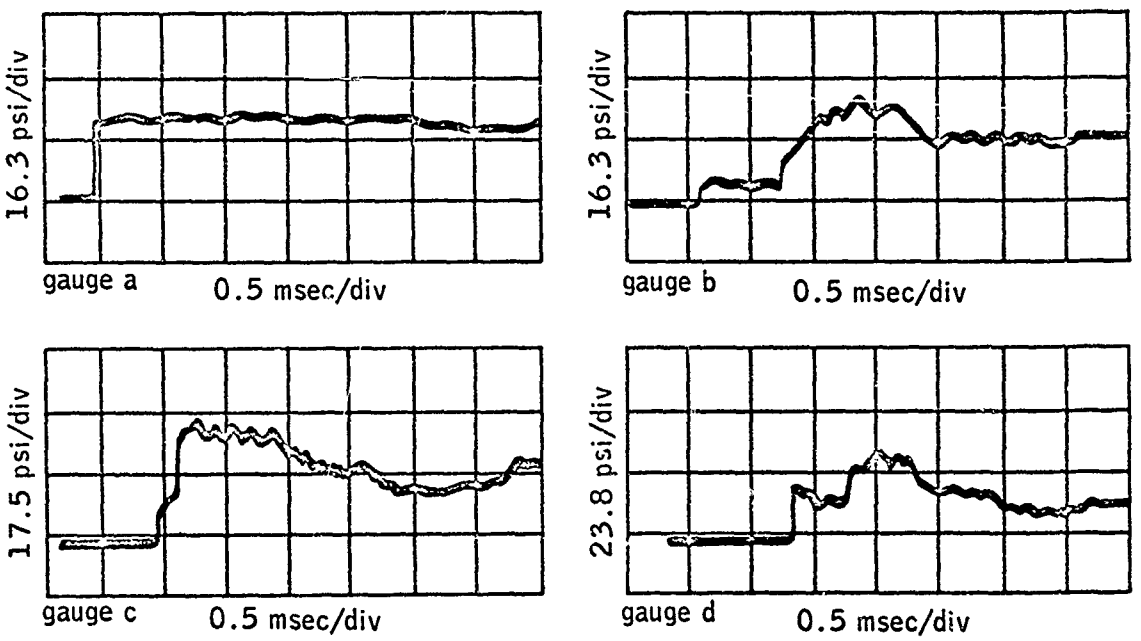


Figure 6

PRESSURE-TIME RECORDS ASSOCIATED WITH
THE DEEP-WITH-OFFSET CHAMBER



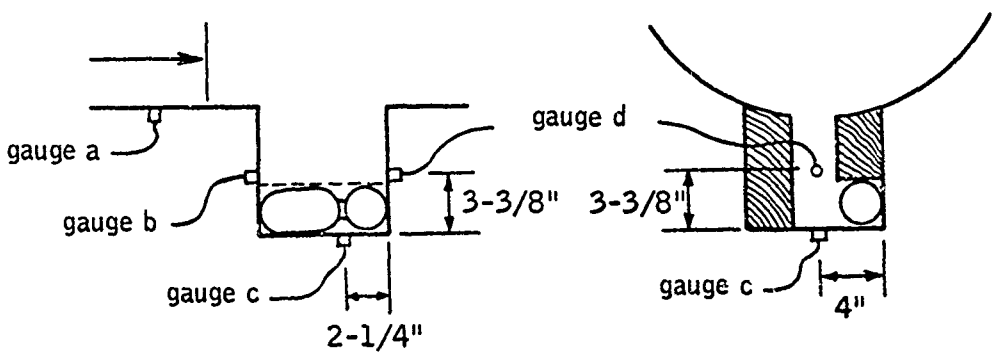


Figure 7

The LD_{50}, in terms of the reflected pressure measured in the deep and deep-with-offset chambers, was 34.6 and 35.9 psi, respectively (Table 3). These LD_{50}'s were not significantly different statistically nor were they different from the LD_{50} incident pressure of 34.9 psi for animals in the shallow chambers.

It should be pointed out here, however, that even though the pressure rose in a stepwise manner in the deep and deep-with-offset chambers with respective times to maximal reflected pressures of near 0.20 and 0.18 msec, the LD_{50} values were not importantly different from those for guinea pigs subjected to near-instantaneous-rising pressures in the long-duration range given in Figure 1. Apparently, time intervals of 0.20 msec or less between arrival of the pressure pulse and the occurrence of maximal pressure did not alter the guinea pig's tolerance to overpressure from what it would be for near-instantaneously rising pressures.

PATHOPHYSIOLOGIC OBSERVATIONS

The Time of Death for Fatally Blasted Animals

Figure 8 gives the cumulative per cent mortality curves for small animals, dogs, and goats. As noted in the figure, the majority of the animals died within 30 minutes after the blast. The averaged curve for the small animals (mice, rats, guinea pigs, rabbits) and for the goats compiled from published [2] and unpublished data were similarly quite steep. On the other hand, the curve for the dogs was less steep since more deaths occurred after 30 minutes. No explanation can be offered at this time for the longer survival times of dogs.

Lung Hemorrhage

It is well known that the gas-containing organs of the body are most affected by air blast[6] – the eardrum being the most sensitive. The lung, however, appears to be the "target" organ or the organ whose injury is critical for the demise of the animal. Characteristically, massive bilateral lung hemorrhage occurs as a result of blast exposure, a fact reflected by an increase in lung weight. Figure 9, from a previous study,[1,6] shows the lung weights as a per cent of body weight for a series of guinea pigs exposed to "sharp"-rising, long-duration overpressures. Note that, on the average, the lung weights for animals surviving longer than 1 hour is lower than those for lethally injured animals.

Similarly Figure 10, from a study in which guinea pigs were

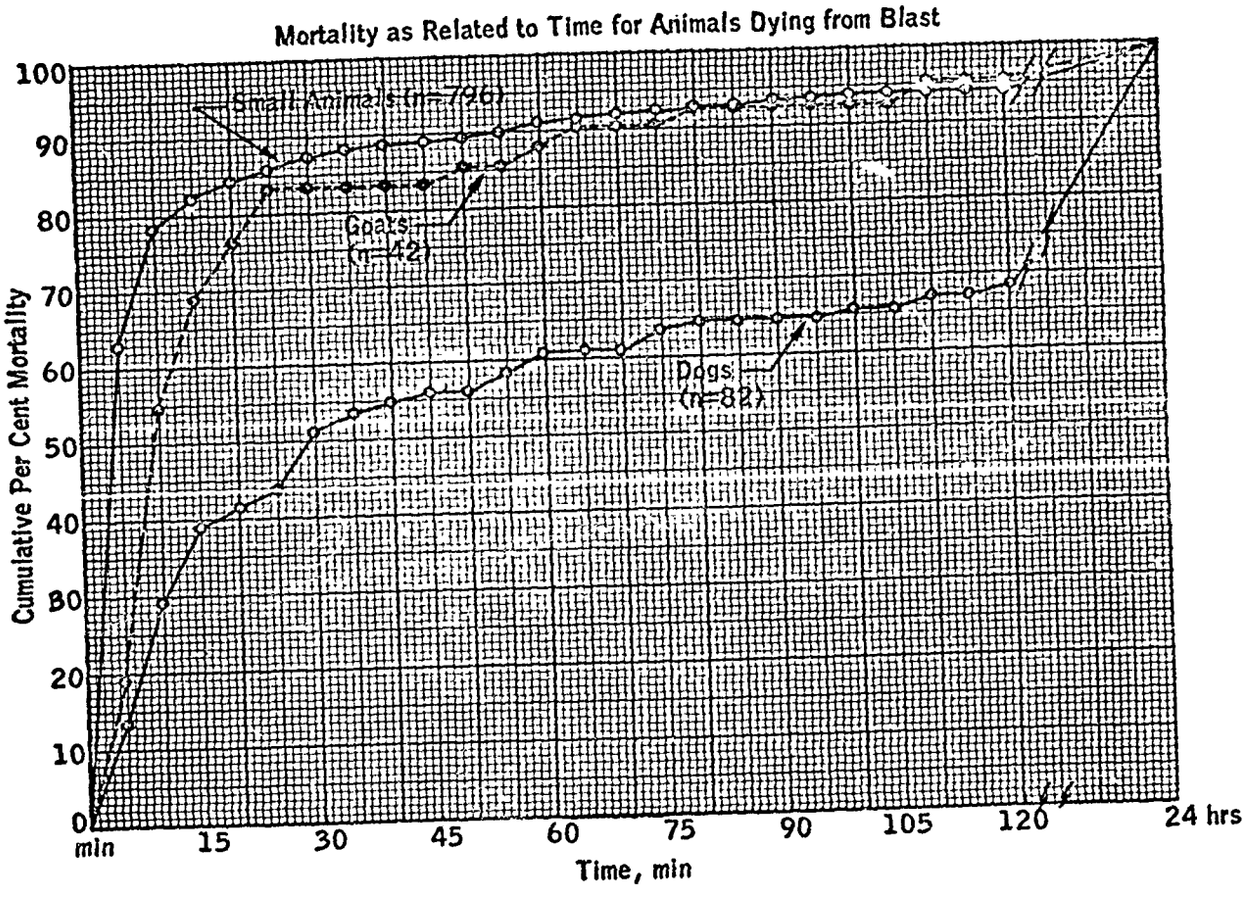

Mortality as Related to Time for Animals Dying from Blast
Cumulative Per Cent Mortality
100
90
80
70
60
50
40
30
20
10
0
Small Animals (n=796)
Goats (n=42)
Dogs (n=82)
min
15
30
45
60
75
90
105
120
24 hrs
Time, min

Figure 8

LUNG WEIGHTS, MORTALITY AND CORONARY
AIR EMBOLISM IN BLASTED GUINEA PIGS

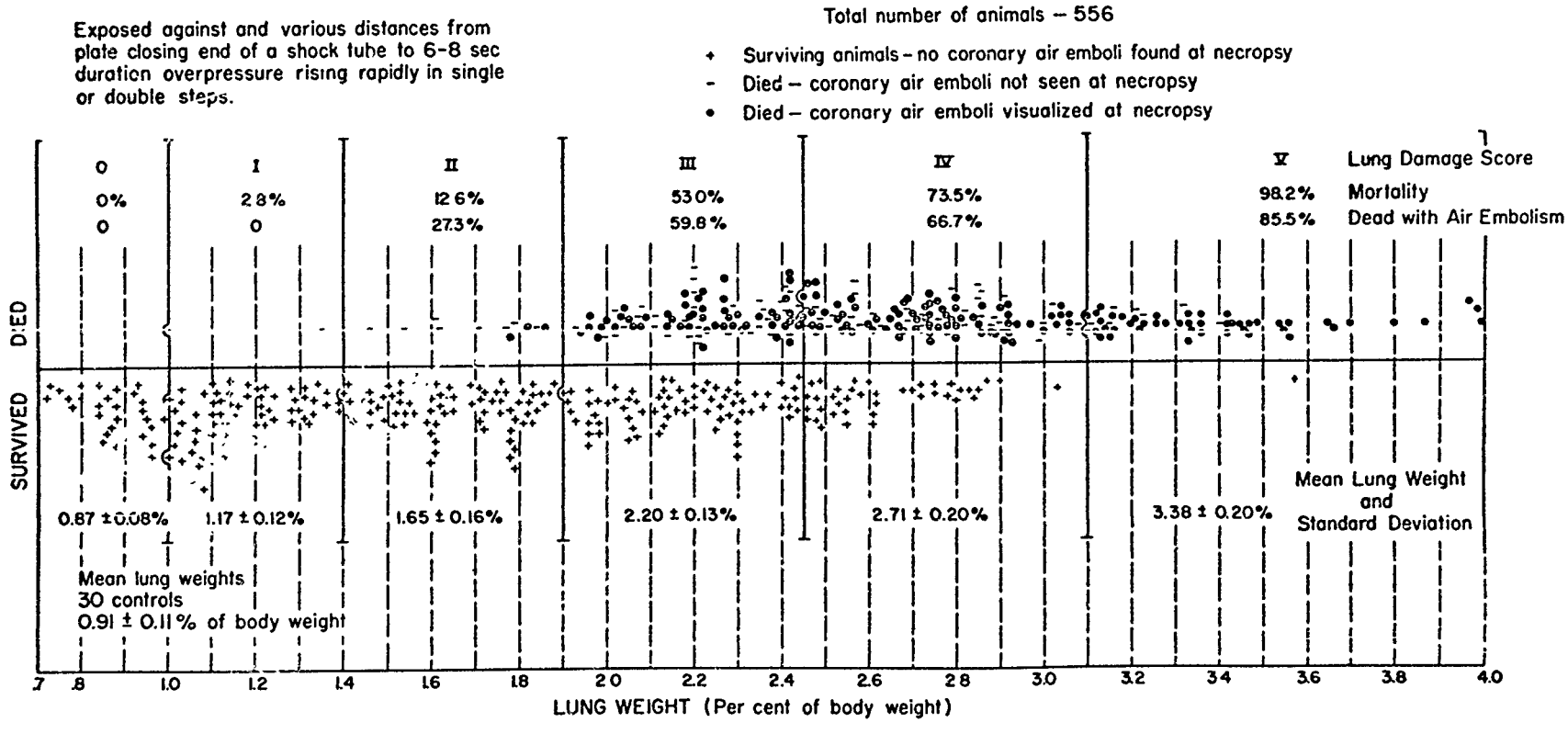


Figure 9

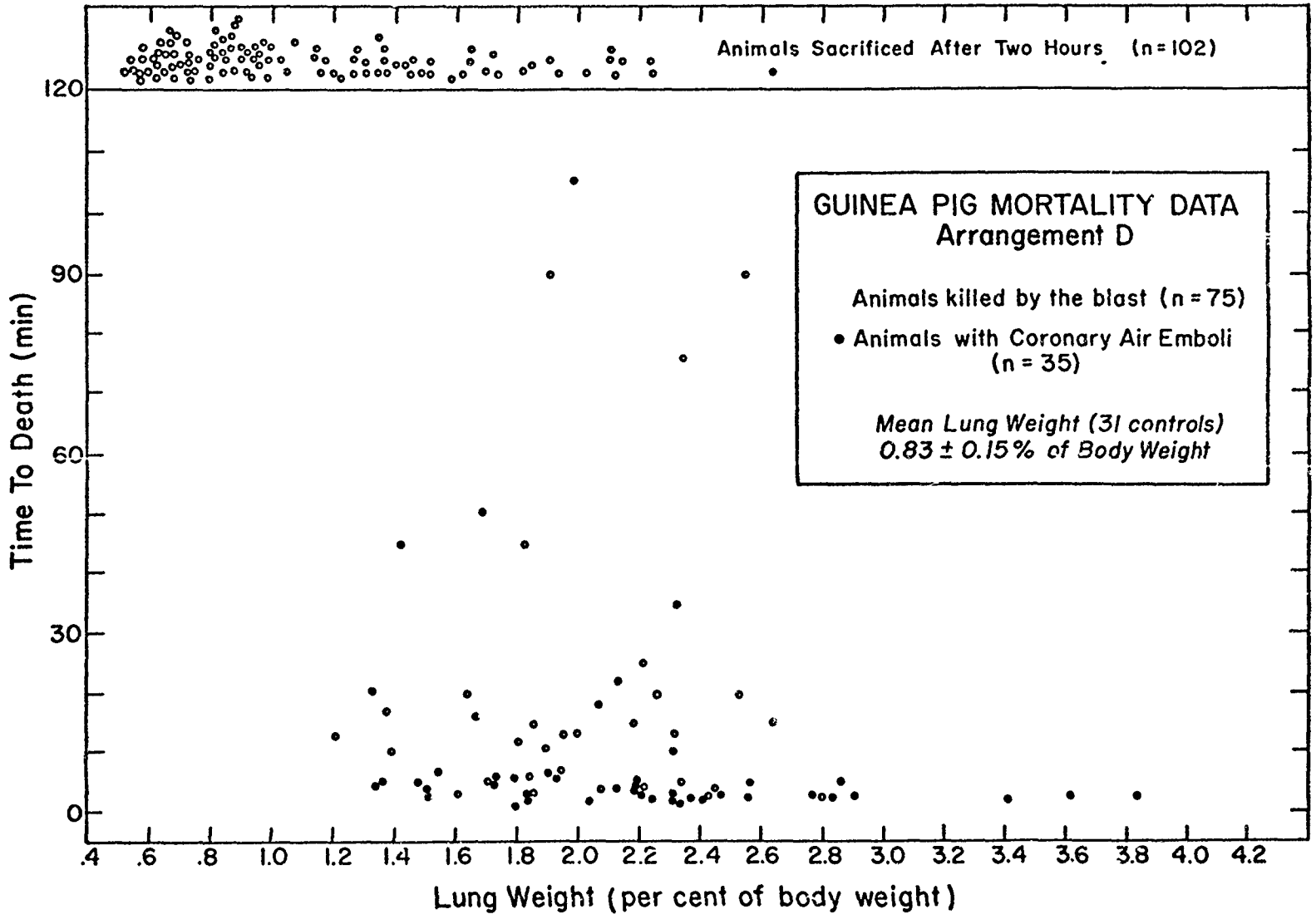

Animals Sacrificed After Two Hours (n=102)
GUINEA PIG MORTALITY DATA
Arrangement D
Animals killed by the blast (n = 75)
• Animals with Coronary Air Emboli (n = 35)
Mean Lung Weight (31 controls)
0.83 ± 0.15% of Body Weight
Time To Death (min)
120
90
60
30
0
.4 .6 .8 1.0 1.2 1.4 1.6 1.8 2.0 2.2 2.4 2.6 2.8 3.0 3.2 3.4 3.6 3.8 4.0 4.2
Lung Weight (per cent of body weight)

Figure 10

exposed to "sharp"-rising overpressures of 3 - 4 msec duration,[2] gives lung weight data in relation to time of death for cases lethally injured compared with those surviving two or more hours.

There are at least two striking additional findings portrayed by Figures 9 and 10. The first is the fact that animals not infrequently die with near-normal values for the per cent lung weights and survivors often yield figures that are two or threefold the normal for the species. The second finding concerns the common occurrence of coronary air emboli in lethally injured animals, particularly those whose death is early. It is clear that any etiologic views of blast death must be consistent with these facts.

Air Emboli

It is believed that air bubbles entering the circulatory system from the damaged lungs explain the experimental findings of arterial air emboli in the coronary and cerebral vessels. Too, the actual mechanism thought responsible for rapid death concerns air emboli and the sequelae therefrom, particularly when the coronary vessels are involved.[2, 5, 6, 11-15] Thus, death with a near-normal lung weight can be explained by early heart failure associated with massive coronary air embolism; i.e., there is not time for lung hemorrhage to become marked. Animals succumbing with massive lung hemorrhage apparently escape immediate coronary failure; they may subsequently die quickly from coronary air embolism involving fairly large vessels or linger and expire either from multiple involvement of small coronary vessels and the early sequelae of coronary air emboli or from continued lung hemorrhage and edema. Seriously injured animals that survive several hours or days face the hazards associated with severe heart and lung damage as well as possible infectious processes centered in the pulmonary tree.

Additional findings are given in Table 4 to supplement the meager data documenting blast-induced air emboli in larger animals.[11-14] Air bubbles were found in autopsy in the coronary arteries of 27 of 42 goats (64 per cent) and in 43 of 82 dogs (52 per cent) killed by the blast. The incidence of air emboli in rabbits and guinea pigs is also given.

Figure 11 shows the occurrence of coronary air emboli as a function of time-to-death in 56 dogs expiring within two hours after exposure to "sharp"-rising overpressures of different durations. There were 43 instances of coronary air embolism, 39 of which occurred in 42 animals whose death was recorded within 30 minutes postshot.

Table 4

THE INCIDENCE OF CORONARY AIR EMBOLI
IN ANIMALS SUBJECTED TO AIR BLAST*

Animal Species	Number Blasted	Number Dead	Number Dead with Coronary Air Emboli
Goats	97	42 (43.3%)	27 (64.3%)
Dogs	204	82 (40.2%)	43 (52.4%)
Rabbits	268	139 (51.9%)	32 (23.0%)
Guinea Pigs	273	156 (57.1%)	66 (42.3%)
	842	419 (49.8%)	168 (40.1%)

*Overpressures rose almost instantaneously and were of various durations.

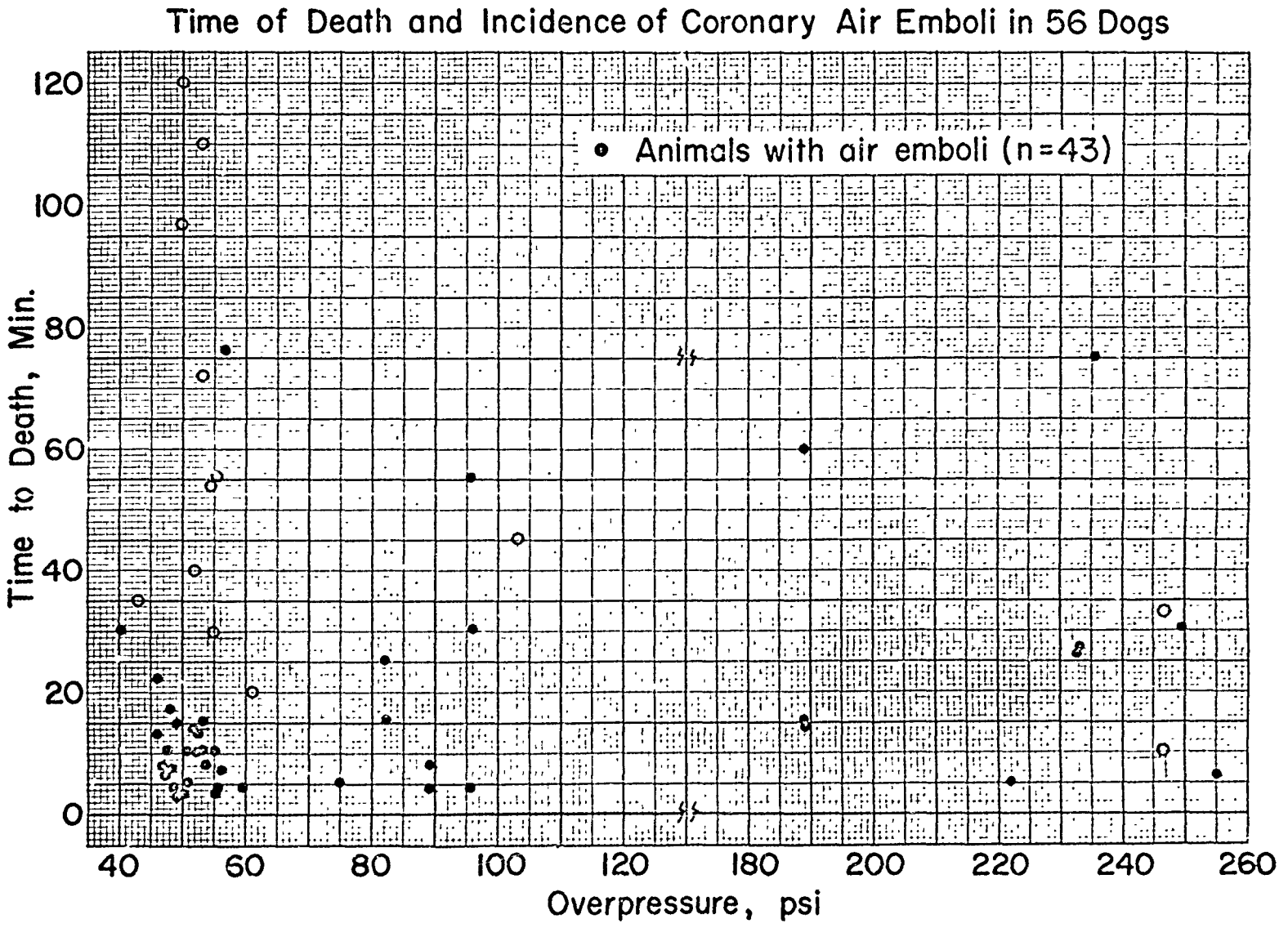

Time of Death and Incidence of Coronary Air Emboli in 56 Dogs
Animals with air emboli (n=43)
Time to Death, Min.
0
20
40
60
80
100
120
40
60
80
100
120
180
200
220
240
260
Overpressure, psi

Figure 11

Figure 12 similarly shows the time of death and occurrence of air emboli among 40 lethally injured goats whose deaths happened within two hours after exposure to "sharp"-rising overpressures of various durations. All the 26 instances of coronary air emboli were noted in 35 animals expiring in the first 30 minutes postshot.

Neuromuscular Signs

Many of the animals killed by blast exhibited symptoms of central nervous system damage such as ataxia, paralysis of the fore and hind limbs, and tonic and clonic convulsive movements. It has been suggested that cerebral air emboli are the cause of these symptoms.[6, 12-15] The animals all appeared to be conscious until very near death, but most of them could not walk – even when they were placed on their feet by the experimenter.

Table 5 summarizes the number of surviving and non-surviving dogs that did or did not walk postshot together with the time periods involved. Of a sample of 55 dogs that died from the blast, only five (9.1 per cent) walked immediately after exposure. Three of the five walked with difficulty (staggered). Of the remaining 50 dogs, only four managed locomotion: one each at 6, 10, 25, and 40 minutes after exposure. Forty-six dogs (83.6 per cent) failed to walk between the time of the blast and their death.

Of the 90 dogs that survived 24 hours, only six did not walk (6.7 per cent) in that period. Thirty animals that could not walk initially after exposure did so between 2 and 210 minutes postshot (Table 5). Fifty-four dogs (60.0 per cent) that survived walked immediately postshot.

Table 6 summarizes the data for goats, which also show a high percentage (68.8) of those lethally blasted being unable to walk.

Also worth considerable emphasis is the fact that animals after significant exposure to blast seem "stunned" and "dazed" by the experience. Characteristically also, they show no signs of pain even when the abdomen or chest is palpated and pressure is applied to the paw; i.e., squeezing the foot pad of dogs or other animals normally elicits prompt withdrawal of the limb. Lethargy and a failure to respond to stimuli occurs even though there is clear indication (in dogs) by head turning and tail wagging that the human voice is heard and appreciated. Except for these and other central nervous system signs and the frequent, but transient appearance of blood at the nose or lips, the blasted animal classically exhibits a misleading normal appearance in that there is little externally to indicate the seriousness of the injury sustained.

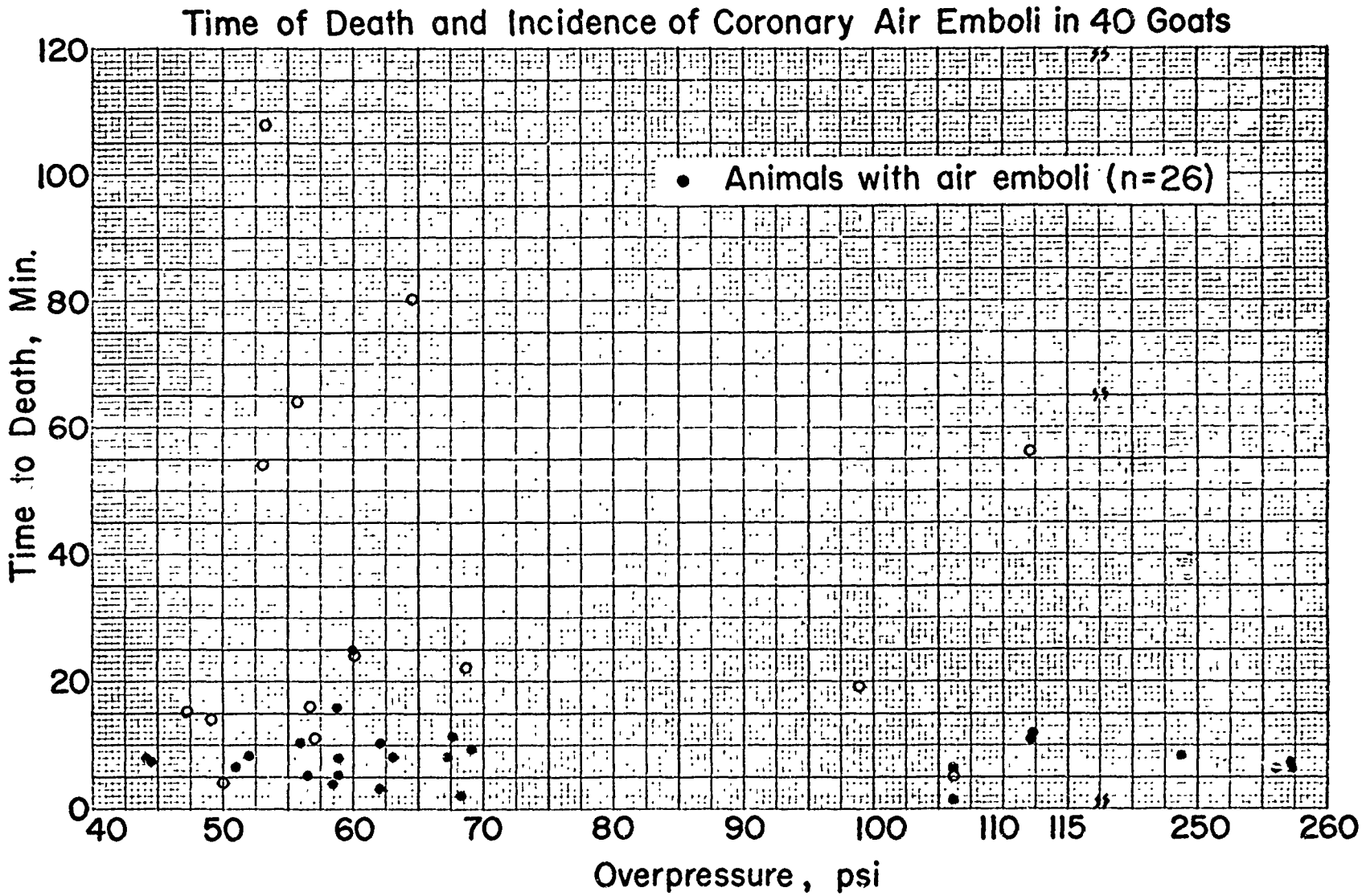

Time of Death and Incidence of Coronary Air Emboli in 40 Goats
Animals with air emboli (n=26)
Time to Death, Min.
0
20
40
60
80
100
120
40
50
60
70
80
90
100
110
115
250
260
Overpressure, psi

Figure 12

Table 5

POSTSHOT LOCOMOTION OF DOGS EXPOSED TO "SHARP"-RISING OVERPRESSURES OF VARIOUS DURATIONS

Remarks	Number of Animals	
	Survivors	Fatalities
Walked immediately postshot	54 (60.0%)	5 (9.1%)
Walked at: 2 min	1	-
6 min	-	1
10 min	12	1
20 min	3	-
25 min	-	1
40 min	-	1
60 min	3	-
90 min	6	-
120 min	1	-
180 min	3	-
210 min	1	-
Failed to walk	6* (6.7%)	46** (83.6%)
Total Number	90	55

*Between test and 24-hour sacrifice.
**Between test and death – mostly short-survival times.

Table 6

POSTSHOT LOCOMOTION OF GOATS EXPOSED TO "SHARP"-RISING OVERPRESSURES OF VARIOUS DURATIONS

Remarks	Number of Animals	
	Survivors	Fatalities
Walked immediately postshot	12 (46.2%)	4 (25.0%)
Walked at: 2 min	1	-
5 min	4	1
10 min	5	-
15 min	1	-
20 min	2	-
60 min	1	-
Failed to walk	-	11* (68.8%)
Total Number	26	16

*Between test and death – mostly short-survival times.

DISCUSSION

Human Air Blast Data

Though the animal data employed in this study for extrapolation to an animal as large as man are not inconsistent with earlier animal investigations in Germany[12-14] and England,[14] it may be said that the validity of information as an indication of the range of mammalian response can and should be enhanced by extending the investigations to include additional numbers of animals both smaller and larger than man; i.e., cats, swine, goats, cattle, horses, chimpanzees, etc.

Also, attention needs be directed to all air-blast data of a quantitative or semi-quantitative nature applying to the human case. Currently, only two bits of significant information in this category are known to the authors. The first concerns a field study in Great Britain involving estimations of overpressures produced by high-explosive bombs for 12 carefully selected instances of human exposure.[16] The scaled overpressures ranged from 170 to 500 - 600 psi with only one fatality related with an overpressure of 450 psi. The durations of the overpressures were not stated, but were probably only a few milliseconds, since the bomb weights for the time period in question ranged from 250 to 1000 lbs (explosive charges of near 125 - 500 lbs).

The second field study in Germany involved the exposure of 13 men in an anti-aircraft gun emplacement to blast from a 918-kg bomb containing 552 kg of explosive.[14] Two deaths occurred at a location where the estimated maximal overpressure was 235 psi resulting from the reflection of an incident shock of 58 psi. The duration of the overpressure was probably between 4 and 6 msec.

It is well to call attention to the fact that earlier tentative estimates of man's tolerance to "fast"-rising overpressures of long duration have been documented.[3, 17-19] The estimates were formulated using then available portions of the data reported in this study. The current estimates as set forth in Figure 3 and Tables 1 and 2 are based upon considerable additional experimentation. They represent, therefore, an extension and updating of previous work. Since the figures suggested as applicable to man are only tentative and because investigative and theoretical explorations continue, one can expect more refined opinions will be forthcoming in the future.

Atypical Wave Forms

All the data employed in the present presentation concerning the estimates of human tolerance refer to "fast"-rising overpressure having ideal or near-ideal wave forms. Though, as pointed out previously, it is known that the tolerance of certain mammals to overpressure increases for a variety of non-ideal pulses of overpressure — particularly if the rising phase of the pressure is delayed — there is practically no useful information to support extrapolation to the 70-kg mammal in this area.[1,6] It is simply necessary to say that the quantitative effects of smooth-rising overpressures and those reaching a maximum in two or more steps must be studied further, especially in larger animals, before any meaningful opinions concerning human tolerance under such circumstances can be set forth.

Threshold Damage

Similar uncertainties exist regarding the minimal overpressure that will produce lung hemorrhage in large as well as small animals. The authors have expressed an opinion that, for "fast"-rising long-duration overpressures, near 15 psi incident overpressure or 6 psi in a geometry where reflection of pressure may occur represents an "educated guess" for the threshold pressure for lung damage in dogs and probably man.[3,17-19] Clearly, it is not possible currently to render even an opinion on what the value is for "fast"-rising short-duration overpressures and for non-ideal wave forms. It is equally clear, however, that this is an important area for future investigations.

Pathophysiology

Though it has not been the intention of this paper to cover any except selected, important portions of the pathophysiological data relevant to the primary effects of air blast, it should be said that damage to the ear, sinuses, and contents of the orbital and abdominal cavities frequently occurs. Those wishing further information are referred to: the excellent work of Clemedson[21] in Sweden which represents by far the most extensive effort that has been directed to the pathophysiology of blast injury; the publications of Benzinger,[13] DeSaga[14] and Rössle[12] whose investigations in Germany give the most complete picture of blast pathology assembled in a single source; and the findings of the group working in Albuquerque[1-6,8,10,11,17-20,22-24] whose papers contain references to other scientists contributing to the field of blast and shock biology.

Blast Protection

It seems appropriate by way of emphasis to make two rather obvious statements that sooner or later interests those who contemplate biological blast effects. The first is the fact that primary blast lesions are often complicated by secondary and tertiary damage from blast-energized debris and accelerative and decelerative loading associated with displacement due to blast shock, pressures and winds. Second, because of the rapid lethality that characterizes exposure to overpressure and decelerative impact[25] as well as the serious nature of penetrating and nonpenetrating wounds of the head and other critical portions of the body,[26] the exploration of all possible means for blast protection is without a doubt indicated.[27,28] In this regard, future refinements of methods and techniques conceived to avoid the damage typical of blast exposure should analytically consider all the blast effects as well as others often associated with modern weapon systems.

GENERAL

Finally, it is somewhat discouraging to note that little definitive can be said concerning the ability of an animal or man to carry on physical or mental activity after serious exposure to air blast. DeSaga,[29] in a study performed for the Air Force of the German Reich, stated an opinion based on animal experimentation and field observations as follows: "Severely as well as slightly injured persons – as soon as they have been exposed to a detonation – can no longer be used as soldiers for military action, duty or operations." Published information also makes it clear that exercise after a significant blast exposure is highly dangerous, and even moving a severely blasted human case is contraindicated.[6]

Be these things as they may, there are certainly uncertainties concerning the relation between the severity of blast damage and the activity of which man is capable. A quotation from a paper by Williams[30] is pertinent: "An incident of interest --------- occurred during the campaign in Norway. Seven men on board one of H. M. ships were in the vicinity of a magazine hatch when the ship blew up. These men managed to reach shore, but one of them felt so ill that he was unable to stand and had to be carried by his comrades to shelter in a school, where the party turned in for the night. The sick man was horrified to wake the next morning and see the remaining six men lying with rather blue faces and to be informed that they were all dead."

REFERENCES

1. Richmond, D. R., R. V. Taborelli, F. Sherping, M. B. Wetherbe, R. T. Sanchez, V. C. Goldizen and C. S. White: Shock tube studies of the effects of sharp-rising, long-duration overpressures on biological systems. AF Document SWR-TM-59-2, pp. 171-194. Proceedings of Third Shock Tube Symposium, March 10-12, 1959, Hq., Air Force Special Weapons Center, ARDC, Kirtland Air Force Base, New Mexico. Also published as USAEC Report TID-6056, Office of Technical Services, Department of Commerce, Washington 25, D. C., March 10, 1959.

2. Richmond, D. R., V. C. Goldizen, V. R. Clare, D. E. Pratt, F. Sherping, R. T. Sanchez, C. C. Fischer and C. S. White: The biologic response to overpressure. III. Mortality in small animals exposed in a shock tube to sharp-rising overpressures of 3 to 4 msec duration. Aerospace Med., 33: 1-27, 1962. Also published as DASA 1242, Defense Atomic Support Agency of the Department of Defense, Washington 25, D. C.

3. Richmond, D. R., V. R. Clare, V. C. Goldizen, D. E. Pratt, R. T. Sanchez and C. S. White: Biological effects of overpressure. II. A shock tube utilized to produce sharp-rising overpressures of 400 milliseconds duration and its employment in biomedical experiments. Aerospace Med., 32: 997-1008, 1961. Also published as DASA 1246, Defense Atomic Support Agency of the Department of Defense, Washington 25, D. C.

4. Richmond, D. R., V. C. Goldizen, V. R. Clare and C. S. White: The overpressure-duration relationship and lethality in small animals. Submitted as a Technical Progress Report, dated September 10, 1962, on Contract DA-49-146-XZ-055 to the Defense Atomic Support Agency of the Department of Defense, Washington 25, D. C.

5. Clare, V. R., D. R. Richmond, V. C. Goldizen, C. C. Fischer, D. E. Pratt, C. S. Gaylord and C. S. White: The effects of shock tube generated step-rising overpressures on guinea pigs located in shallow chambers oriented side-on and end-on to the incident shock. Submitted as a Technical Progress Report, dated May 31, 1962, on Contract DA-49-146-XZ-055 to the Defense Atomic Support Agency of the Department of Defense, Washington 25, D. C.

6. White, C. S. and D. R. Richmond: Blast biology. Submitted as a Technical Progress Report, dated September 18, 1959, on Contract AT(29-1)-1242 to The Division of Biology and Medicine, United States Atomic Energy Commission. USAEC Report TID-5764, AEC Technical Information Service Extension, Oak Ridge, Tennessee.

7. Schardin, H. and O. Wünsche: Versuche an kleintieren zur bestimmung der druckstosstodlichkeitsgrenze. Bericht V 17, Deutsche Versuchsanstalt für Luftfahrt e. V., January 1958.

8. Richmond, D. R., D. E. Pratt and C. S. White: Orbital "blow-out" fractures in dogs produced by air blast. Submitted as a Technical Progress Report, dated April 10, 1962, on Contract DA-49-146-XZ-055 to the Defense Atomic Support Agency of the Department of Defense, Washington 25, D. C.

9. Froboese, M. and O. Wünsche: Todlichkeitsgrenzen für albino-ratten bei luftstossbelastung in abhängigkeit von der strossrichtung und druckverlaufsform. Rapport Bericht, Band I: Text, 2/59, French-German Research Institute of St. Louis, France, June 30, 1959.

10. Richmond, D. R., M. B. Wetherbe, R. V. Taborelli, T. L. Chiffelle and C. S. White: The biologic response to overpressure. I. Effects on dogs of five to ten-second duration overpressures having various times of pressure rise. J. Aviat. Med., 28: 447-460, 1957.

11. Richmond, D. R., V. R. Clare and C. S. White: The tolerance of guinea pigs to air blast when mounted in shallow, deep, and deep-with-offset chambers on a shock tube. Technical Progress Report on Contract DA-49-146-XZ-055 to the Defense Atomic Support Agency of the Department of Defense, Washington 25, D. C. (In preparation)

12. Rössle, R.: Pathology of blast effects. Chap. XIV-C, pp. 1260-1273, Vol. II, German Aviation Medicine, World War II, U. S. Government Printing Office, Washington 25, D. C., 1950.

13. Benzinger, T.: Physiological effects of blast in air and water. Chap. XIV-B, German Aviation Medicine, World War II, Vol. II, pp. 1225-1259, U. S. Government Printing Office, Washington 25, D. C., 1950.

14. DeSaga, Hans: Blast injuries. Chap. XIV-D, German Aviation Medicine, World War II, Vol. II, pp. 1274-1293, U. S. Government Printing Office, Washington 25, D. C., 1950.

15. Clemedson, Carl-Johan and H. Hultman: Air embolism and the cause of death in blast injury. Military Surgeon, 114: 424-437, 1954.

16. Fisher, R. B., P. L. Krohn and S. Zuckerman: The relationship between body size and the lethal effects of blast. Report R. C. 284, Ministry of Home Security, Oxford, England, December 10, 1941.

17. White, C. S., I. G. Bowen, D. R. Richmond and R. L. Corsbie: Comparative nuclear effects of biomedical interest. Civil Effects Test Operations, USAEC Report CEX-58.8, January 12, 1961, Office of Technical Services, Department of Commerce, Washington 25, D. C.

18. White, C. S.: Biological effects of blast. Presented before the Armed Forces Medical Symposium, Field Command, Defense Atomic Support Agency, Sandia Base, Albuquerque, New Mexico, November 28, 1961. Submitted as a Technical Progress Report on Contract DA-49-146-XZ-055 to the Defense Atomic Support Agency in December, 1961. Also published as DASA 1271, Defense Atomic Support Agency of the Department of Defense, Washington 25, D. C.

19. White, C. S., D. R. Richmond, I. G. Bowen, T. L. Chiffelle and R. V. Taborelli: The biological effects of blast from bombs — Summary for period 1960-1962, July 1, 1962. Submitted to the Defense Atomic Support Agency of the Department of Defense, Washington 25, D. C., July 12, 1962.

20. Goldizen, V. C., D. R. Richmond, T. L. Chiffelle, I. G. Bowen and C. S. White: Missile studies with a biological target. Operation Plumbbob Report, WT-1470, AEC Civil Effects Test Group, January 23, 1961. Office of Technical Services, Department of Commerce, Washington 25, D. C.

21. Clemedson, Carl-Johan: An experimental study on air blast injuries. Acta Physiol. Scand., 18, Supplement 61: 1-200, 1949.

22. White, C. S., T. L. Chiffelle, D. R. Richmond, W. H. Lockyear, I. G. Bowen, V. C. Goldizen, H. W. Merideth, D. E. Kilgore, B. B. Longwell, J. T. Parker, F. Sherping and M. E. Cribb: Biological effects of pressure phenomena occurring inside protective shelters following a nuclear detonation. Operation Teapot Report, WT-1179, October 28, 1957, Office of Technical Services, Department of Commerce, Washington 25, D. C.

23. Richmond, D. R., R. V. Taborelli, I. G. Bowen, T. L. Chiffelle, F. G. Hirsch, B. B. Longwell, J. G. Riley, C. S. White, F. Sherping, V. C. Goldizen, J. D. Ward, M. B. Wetherbe, V. R. Clare, M. L. Kuhn and R. T. Sanchez: Blast biology – A study of the primary and tertiary effects of blast in open underground protective shelters. Operation Plumbbob Report, WT-1467, June 30, 1959, Office of Technical Services, Department of Commerce, Washington 25, D. C.

24. Roberts, J. E., C. S. White and T. L. Chiffelle: Effects of overpressures in group shelters on animals and dummies, Part I, Project 23.15, Operation Upshot-Knothole, Report WT-798, September 1953, AEC Technical Information Service, Oak Ridge, Tennessee.

25. Richmond, D. R., I. G. Bowen and C. S. White: Tertiary blast effects: Effects of impact on mice, rats, guinea pigs and rabbits. Aerospace Med., 32: 789-805, 1961. Also published as DASA 1245, Defense Atomic Support Agency of the Department of Defense, Washington 25, D. C.

26. Coates, Jr., J. B. and J. C. Beyer: Wound ballistics. Office of the Surgeon General, Department of the Army, U. S. Government Printing Office, Washington 25, D. C., 1962.

27. Beyer, J. C., Enos, W. F., and R. H. Holmes: Personnel protective armor. Wound Ballistics, Chapter XI. Office of the Surgeon General, Department of the Army, U. S. Government Printing Office, Washington 25, D. C., 1962.

28. Herget, C. M., G. B. Coe, and J. C. Beyer: Wound ballistics and body armor in Korea. Wound Ballistics, Chapter XII. Office of the Surgeon General, Department of the Army, U. S. Government Printing Office, Washington 25, D. C., 1962.

29. DeSaga, H.: Experimentelle untersuchungen der Luftstoszwirkung. Forschungsbericht 15/43. Mitteilungeben aus dem Gebiet der Luftfabrtmedizin. Herausgegeben von Inspektein des Sanitätswesens der Luftwaffe, 1943.

30. Williams, E. R. P.: Blast effects in warfare. Brit. Jour. Surg., 30: 38-49, 1942.

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Attn: Col. S. W. Cavender, Surgeon

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The Dikewood Corporation
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Douglas Aircraft Company, Inc.
El Segundo Division
El Segundo, California
Attn: Mr. Harvey Glassner
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Federal Aviation Agency
Washington 25, D.C.
Attn: Dr. James L. Goddard, Civil Air Surgeon

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Mr. Kenneth Kaplan
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Lockheed Aircraft Corporation 3
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Lockheed Aircraft Corporation
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The Martin Company 1
Denver, Colorado
Attn: Dr. James G. Gaume, Chief, Space Medicine

McDonnel Aircraft Company
Lambert Field
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Mr. Bert North

National Aeronautics and Space Administration 1
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Naval Medical Research Institute 1
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Department of the Navy 1
Bureau of Medicine & Surgery
Washington 25, D.C.
Attn: Capt. G. J. Duffner, Director, Submarine Medical Division

North American Aviation 4
International Airport
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Attn: Scott Crossfield
Dr. Toby Freedman, Flight Surgeon
Mr. Fred A. Payne, Manager Space Planning,
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Republic Aviation Corporation 3
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System Development Corporation 1
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University of New Mexico 1
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U. S. Naval Ordnance Laboratory 2
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